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NEWS 4	May 12	Polymer links for the POLYLINK command completed in REGISTRY
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=> s clotting (3A) (factor (w) (II or VII or IX or X))
  13 FILES SEARCHED...
  24 FILES SEARCHED...
  32 FILES SEARCHED...
  42 FILES SEARCHED...
  52 FILES SEARCHED...
  59 FILES SEARCHED...
  64 FILES SEARCHED...
  78 FILES SEARCHED...
  94 FILES SEARCHED...
L1         7347 CLOTTING (3A) (FACTOR (W) (II OR VII OR IX OR X))

=> s L1 (6A) ((dominant negative) or inactive)
  28 FILES SEARCHED...
  58 FILES SEARCHED...
  72 FILES SEARCHED...
L2            5 L1 (6A) ((DOMINANT NEGATIVE) OR INACTIVE)

=> d l2 1-5 bib ab

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AN    1985:15038456   BIOTECHNO
TI    Structure and function of factor IX: Defects in haemophilia B
AU    McGraw R.A.; Davis L.M.; Lundblad R.L.; et al.
CS    Department of Pathology, University of North Carolina, Chapel Hill, NC
```

27514, United States.
SO Clinics in Haematology, (1985), 14/2 (359-383)
CODEN: CLHMB3
DT Journal; Article
CY United Kingdom
LA English
AB The genetics of haemophilia B and the structure-function relationships of factor IX interactions with cofactors and substrates have been reviewed. Emphasis has been placed on contributions to our understanding made by analysis of variants. Amino acid substitutions at or near the site of activation lead to **inactive** factor IX or to **factor IX** species with decreased **clotting** activity. Release of the activation peptide is necessary for optimal interaction of factor IX with its cofactors and substrates. Abnormalities in the calcium binding region, whether Gla dependent or independent, also decrease clotting activity. The defects in haemophilia B(m) variants somehow affect factor VII-tissue factor interactions with factor X. Other mutations may affect the facotor IX heavy chain, probably at or near the active site. Amino acid substitutions may cause conformational changes in factor IX that interfere with other interactions such as with antithrombin III and factor VIII. Recombinant DNA techniques have been employed to analyse normal and abnormal factor IX genes. DNA sequence analysis of factor IX cDNA clones revealed the primary structure of the mature protein and a predicted leader peptide. Knowledge of the primary sequence of factor IX allowed identification of the specific defect in the factor IX (Chapel Hill) variant. Analysis of normal factor IX genomic clones has determined that the 35 kb gene is composed of eight coding exons and seven interventing sequences. Sequence analysis of the CRM.sup.+ variants will identify mutations disrupting the normal interactions of factor IX. Southern analysis of CRM.sup.- variants has revealed gross factor IX gene deletions in some cases. Such deletions have been employed for carrier deletion in some families. Restriction fragment length polymorphisms in the factor IX gene have also proven useful for carrier identification. Manipulations of the cloned factor IX gene to make specific mutations in vitro and improvements in the technology for expression of deliberately modified genes will further elucidate the relationships between factor IX structure and function.

L2 ANSWER 2 OF 5 EMBASE
on STN
AN 85188456 EMBASE
DN 1985188456
TI Structure and function of factor IX: Defects in haemophilia B.
AU McGraw R.A.; Davis L.M.; Lundblad R.L.; et al.
CS Department of Pathology, University of North Carolina, Chapel Hill, NC 27514, United States
SO Clinics in Haematology, (1985) 14/2 (359-383).
CODEN: CLHMB3
CY United Kingdom
DT Journal
FS 037 Drug Literature Index
025 Hematology
022 Human Genetics
029 Clinical Biochemistry
LA English
AB The genetics of haemophilia B and the structure-function relationships of factor IX interactions with cofactors and substrates have been reviewed. Emphasis has been placed on contributions to our understanding made by analysis of variants. Amino acid substitutions at or near the site of activation lead to **inactive** factor IX or to **factor IX** species with decreased **clotting** activity. Release of the activation peptide is necessary for optimal interaction of factor IX with its cofactors and substrates. Abnormalities in the calcium binding

region, whether Gla dependent or independent, also decrease clotting activity. The defects in haemophilia B(m) variants somehow affect factor VII-tissue factor interactions with factor X. Other mutations may affect the facotor IX heavy chain, probably at or near the active site. Amino acid substitutions may cause conformational changes in factor IX that interfere with other interactions such as with antithrombin III and factor VIII. Recombinant DNA techniques have been employed to analyse normal and abnormal factor IX genes. DNA sequence analysis of factor IX cDNA clones revealed the primary structure of the mature protein and a predicted leader peptide. Knowledge of the primary sequence of factor IX allowed identification of the specific defect in the factor IX (Chapel Hill) variant. Analysis of normal factor IX genomic clones has determined that the 35 kb gene is composed of eight coding exons and seven interventing sequences. Sequence analysis of the CRM+ variants will identify mutations disrupting the normal interactions of factor IX. Southern analysis of CRM- variants has revealed gross factor IX gene deletions in some cases. Such deletions have been employed for carrier deletion in some families. Restriction fragment length polymorphisms in the factor IX gene have also proven useful for carrier identification. Manipulations of the cloned factor IX gene to make specific mutations in vitro and improvements in the technology for expression of deliberately modified genes will further elucidate the relationships between factor IX structure and function.

L2 ANSWER 3 OF 5 MEDLINE on STN
AN 86003005 MEDLINE
DN PubMed ID: 3899439
TI Structure and function of factor IX: defects in haemophilia B.
AU McGraw R A; Davis L M; Lundblad R L; Stafford D W; Roberts H R
NC HL 06350 (NHLBI)
HL 07149 (NHLBI)
SO Clinics in haematology, (1985 Jun) 14 (2) 359-83. Ref: 64
Journal code: 0331547. ISSN: 0308-2261.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 198511
ED Entered STN: 19900321
Last Updated on STN: 19990129
Entered Medline: 19851113
AB The genetics of haemophilia B and the structure-function relationships of factor IX interactions with cofactors and substrates have been reviewed. Emphasis has been placed on contributions to our understanding made by analysis of variants. Amino acid substitutions at or near the site of activation lead to **inactive** factor IX or to **factor IX** species with decreased **clotting** activity. Release of the activation peptide is necessary for optimal interaction of factor IX with its cofactors and substrates. Abnormalities in the calcium binding region, whether Gla independent or dependent, also decrease clotting activity. The defects in haemophilia Bm variants somehow affect factor VII-tissue factor interactions with factor X. Other mutations may affect the factor IX heavy chain, probably at or near the active site. Amino acid substitutions may cause conformational changes in factor IX that interfere with other interactions such as with antithrombin III and factor VIII. Recombinant DNA techniques have been employed to analyse normal and abnormal factor IX genes. DNA sequence analysis of factor IX cDNA clones revealed the primary structure of the mature protein and a predicted leader peptide. Knowledge of the primary sequence of factor IX allowed identification of the specific defect in the factor IX Chapel Hill variant. Analysis of normal factor IX genomic clones has determined that the 35 kb gene is composed of eight coding exons and seven intervening sequences. Sequence analysis of the CRM+ variants will identify mutations

disrupting the normal interactions of factor IX. Southern analysis of CRM- variants has revealed gross factor IX gene deletions in some cases. Such deletions have been employed for carrier deletion in some families. Restriction fragment length polymorphisms in the factor IX gene have also proven useful for carrier identification. Manipulations of the cloned factor IX gene to make specific mutations in vitro and improvements in the technology for expression of deliberately modified genes will further elucidate the relationships between factor IX structure and function.

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AN 2000:4832 PHIN
DN C00656444
DED 6 Mar 2000
TI Small implant could provide new treatment for haemophiliacs
SO Clinica (2000) No. 898 p17
DT Newsletter
FS FULL

L2 ANSWER 5 OF 5 INVESTEXT COPYRIGHT 2004 TFS on STN

AN 95:465942 INVESTEXT(tm) REPORT NUMBER:1594142
PGNO PAGE 3 OF 35
DN 1594142
TI COR Therapeutics - Company Report
AU Lenstra, R., et al
CS SMITH BARNEY; NEW YORK (STATE OF)
CSR MID-ATLANTIC/MIDDLE ATLANTIC REGION; UNITED STATES OF AMERICA; NORTH AMERICA
CSTY Financial center investment bank-broker
PD 12 May 1995
DT COMPANY REPORT
FS Text Page; COMPANY REPORT
WC 414

=>
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=> s blood (5A) (factor (w) (II or VII or IX or X))
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27 FILES SEARCHED...
39 FILES SEARCHED...
52 FILES SEARCHED...
61 FILES SEARCHED...
65 FILES SEARCHED...
82 FILES SEARCHED...
96 FILES SEARCHED...
L3 13898 BLOOD (5A) (FACTOR (W) (II OR VII OR IX OR X))

=> s l3 (8A) ((DOMINANT NEGATIVE) OR INACTIVE)
33 FILES SEARCHED...
65 FILES SEARCHED...
97 FILES SEARCHED...
L4 31 L3 (8A) ((DOMINANT NEGATIVE) OR INACTIVE)

=> s l4 and (treat or treatment)
13 FILES SEARCHED...
32 FILES SEARCHED...
53 FILES SEARCHED...
64 FILES SEARCHED...
92 FILES SEARCHED...
L5 27 L4 AND (TREAT OR TREATMENT)

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L6 20 DUPLICATE REMOVE L5 (7 DUPLICATES REMOVED)

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AN 2004:95303 USPATFULL
TI Agents affecting thrombosis and hemostasis
IN Wolf, David L., Davis, CA, UNITED STATES
Sinha, Uma, San Francisco, CA, UNITED STATES
PA COR Therapeutics, Inc. (U.S. corporation)
PI US 2004072757 A1 20040415
AI US 2003-712332 A1 20031113 (10)
RLI Continuation of Ser. No. US 2000-671346, filed on 27 Sep 2000, PENDING Continuation of Ser. No. US 1999-362207, filed on 28 Jul 1999, ABANDONED Continuation of Ser. No. US 1998-16403, filed on 30 Jan 1998, GRANTED, Pat. No. US 5968897 Division of Ser. No. US 1995-469301, filed on 6 Jun 1995, GRANTED, Pat. No. US 5837679 Division of Ser. No. US 1994-268003, filed on 29 Jun 1994, GRANTED, Pat. No. US 5583107 Continuation-in-part of Ser. No. US 1994-249777, filed on 26 May 1994, GRANTED, Pat. No. US 5597799 Continuation of Ser. No. US 1991-808329, filed on 16 Dec 1991, ABANDONED Continuation-in-part of Ser. No. US 1990-578646, filed on 4 Sep 1990, GRANTED, Pat. No. US 5278144
DT Utility
FS APPLICATION
LREP MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, CAMBRIDGE, MA, 02139
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 15 Drawing Page(s)
LN.CNT 2150
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Analogs of blood factors which are transiently inactive are useful in **treatment** of diseases characterized by thrombosis. In addition, modified forms of activated blood factors that generate the active blood factor in serum but have extended half-lives are useful in treating hemophilic conditions. These modified forms of the blood factor may be acylated forms which are slowly deacylated in vivo.

L6 ANSWER 2 OF 20 USPATFULL on STN
AN 2004:57926 USPATFULL
TI Liquid composition of modified factor VII polypeptides
IN Hansen, Birthe Lykkegaard, Vaerlose, DENMARK
Jensen, Michael Bech, Allerod, DENMARK
Kornfelt, Troels, Virum, DENMARK
PI US 2004043933 A1 20040304
AI US 2003-602340 A1 20030623 (10)
RLI Continuation-in-part of Ser. No. WO 2002-DK894, filed on 20 Dec 2002, UNKNOWN
PRAI DK 2001-1948 20011221

DK 2001-1949 20011221
US 2002-346888P 20020107 (60)
US 2002-346399P 20020107 (60)
DT Utility
FS APPLICATION
LREP Reza Green, Esq., Novo Nordisk Pharmaceuticals, Inc., 100 College Road West, Princeton, NJ, 08540
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1155
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a liquid, aqueous composition, comprising (i) a modified factor VII polypeptide; (ii) an agent suitable for keeping pH in the range of from about 4.0 to about 8.0; (iii) an antioxidant; and (iv) an agent selected from the list of: a calcium salt, a magnesium salt, or a mixture thereof.

L6 ANSWER 3 OF 20 USPATFULL on STN
AN 2004:205829 USPATFULL
TI Stable blood coagulation inhibitor-free factor vii preparation and method for preparing same
IN Matthiessen, Peter, Vienna, AUSTRIA
Turecek, Peter, Klosterneuburg, AUSTRIA
Schwarz, Hans-Peter, Vienna, AUSTRIA
PA Baxter Aktiengesellschaft, Vienna, AUSTRIA (non-U.S. corporation)
PI US 6777390 B1 20040817
WO 9966031 19991223
AI US 2001-719945 20010220 (9)
WO 1999-AT154 19990614
PRAI AT 1998-1043 19980617
DT Utility
FS GRANTED
EXNAM Primary Examiner: Carlson, Karen Cochrane; Assistant Examiner: Snedden, Sheridan
LREP Heller Ehrman White & McAuliffe LLP
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 624
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Stable pharmaceutical preparations containing blood coagulation Factor VII is disclosed. The pharmaceutical preparations containing blood coagulation Factor VII are free of coagulation inhibitors and are stable over a wide range of environmental conditions. Also provided are blood coagulation Factor VII preparations having a minimum activity of 50 Units/mg of protein that contain less than 5% activated blood coagulation Factor VII (Factor VIIa). The blood coagulation Factor VII containing preparations may also contain other blood coagulation factors and are free from detectable transmissible human pathogens.

L6 ANSWER 4 OF 20 USPATFULL on STN
AN 2003:258328 USPATFULL
TI Factor X analogues having a modified protease cleavage site
IN Himmelspach, Michele, Leopoldsdorf, AUSTRIA
Schlokat, Uwe, Orth/Donau, AUSTRIA
Dorner, Friedrich, Vienna, AUSTRIA
Fisch, Andreas, St. Gallen, SWITZERLAND
Eibl, Johann, Vienna, AUSTRIA
PI US 2003181381 A1 20030925
AI US 2003-407123 A1 20030404 (10)
RLI Division of Ser. No. US 1999-367791, filed on 12 Nov 1999, GRANTED, Pat. No. US 6573071 A 371 of International Ser. No. WO 1998-AT45, filed on 27

Feb 1998, UNKNOWN
PRAI AT 1997-335 19970227
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
CLMN Number of Claims: 51
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 2349
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Factor X analogues having a modification in the region of the natural Factor Xa activation cleavage site, said modification representing a processing site of a protease not naturally cleaving in this region of the Factor X sequence, preparations containing the Factor X analogues according to the invention, and processes for the preparation thereof are described.

L6 ANSWER 5 OF 20 USPATFULL on STN
AN 2003:200929 USPATFULL
TI Factor X deletion mutants and analogues thereof
IN Himmelspach, Michele, Leopoldsdorf, AUSTRIA
Pfleiderer, Michael, Darmstadt, GERMANY, FEDERAL REPUBLIC OF
Falkner, Falko-Guenter, Orth/Donau, AUSTRIA
Eibl, Johann, Vienna, AUSTRIA
Dorner, Friedrich, Vienna, AUSTRIA
Schlokat, Uwe, Orth/Donau, AUSTRIA
PI US 2003138914 A1 20030724
AI US 2003-348504 A1 20030121 (10)
RLI Division of Ser. No. US 1999-367777, filed on 18 Nov 1999, GRANTED, Pat. No. US 6562598 A 371 of International Ser. No. WO 1998-AT46, filed on 27 Feb 1998, UNKNOWN
PRAI AT 1997-336 19970227
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
CLMN Number of Claims: 45
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 2232
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Factor XΔ analogues having a deletion of amino acids Arg180 to Arg234 and a modification in the region of the amino acid sequence between Gly173 and Arg179, preparations containing these factor XΔ analogues, and processes for the preparation thereof are described.

L6 ANSWER 6 OF 20 USPATFULL on STN
AN 2003:148878 USPATFULL
TI Factor X analogues with a modified protease cleavage site
IN Himmelspach, Michele, Leopoldsdorf, AUSTRIA
Schlokat, Uwe, Orth/Donau, AUSTRIA
Dorner, Friedrich, Vienna, AUSTRIA
Fisch, Andreas, St. Gallen, SWITZERLAND
Eibl, Johann, Vienna, AUSTRIA
PA Baxter Aktiengesellschaft, Vienna, AUSTRIA (non-U.S. corporation)
PI US 6573071 B1 20030603
WO 9838317 19980903
AI US 1999-367791 19991112 (9)
WO 1998-AT45 19980227
PRAI AT 1997-335 19970227
DT Utility
FS GRANTED

EXNAM Primary Examiner: Low, Christopher S. F.; Assistant Examiner: Schnizer, Holly
LREP Townsend and Townsend and Crew, L.L.P.
CLMN Number of Claims: 64
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 13 Drawing Page(s)
LN.CNT 2472
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Factor X analogues having a modification in the region of the natural Factor Xa activation cleavage site, said modification representing a processing site of a protease not naturally cleaving in this region of the Factor X sequence, preparations containing the Factor X analogues according to the invention, and processes for the preparation thereof are described.

L6 ANSWER 7 OF 20 USPATFULL on STN
AN 2003:129813 USPATFULL
TI Factor X deletion mutants and analogues thereof
IN Himmelspach, Michele, Leopoldsdorf, AUSTRIA
Pfleiderer, Michael, Darmstadt, GERMANY, FEDERAL REPUBLIC OF
Falkner, Falko-Guenter, Orth/Donau, AUSTRIA
Eibl, Johann, Vienna, AUSTRIA
Dorner, Friedrich, Vienna, AUSTRIA
Schlokat, Uwe, Orth/Donau, AUSTRIA
PA Baxter Aktiengesellschaft, Vienna, AUSTRIA (non-U.S. corporation)
PI US 6562598 B1 20030513
WO 9838318 19980903
AI US 1999-367777 19991118 (9)
WO 1998-AT46 19980227
PRAI AU 1997-336 19970227
DT Utility
FS GRANTED
EXNAM Primary Examiner: Carlson, Karen Cochrane; Assistant Examiner: Snedden, Sheridan
LREP Townsend and Townsend and Crew LLP
CLMN Number of Claims: 56
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2334
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Factor XΔ analogues are provided, as well as pharmaceutical preparations containing such analogues and methods of preparing such analogues. The factor XΔ analogues have a deletion of the amino acids Arg180 to Arg234 and a modification in the region of the amino acid sequence between Gly173 and Arg179 of the factor X amino acid sequence. Such analogues can include a processing site not normally present in factor X, thus allowing for selective conversion of the analogue to an active form. The analogues and preparations have utility in the **treatment** of a number of blood coagulation disorders.

L6 ANSWER 8 OF 20 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 1
AN 2003:68367 BIOSIS
DN PREV200300068367
TI Methods for treating hemophilia A and B and AIDS and devices used therein.
AU Pollard, Harvey B. [Inventor, Reprint Author]; Pollard, Bette S. [Inventor]
CS 1008 Lamplighter La., Potomac, MD, 20854, USA
PI US 6491655 December 10, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (Dec 10 2002) Vol. 1265, No. 2. http://www.uspto.gov/web/menu/patdata.html . e-file.
ISSN: 0098-1133 (ISSN print).

DT Patent
LA English
ED Entered STN: 29 Jan 2003
Last Updated on STN: 29 Jan 2003
AB The present invention provides a method for treating Hemophilia A or B which comprises implanting in fluid communication with the bloodstream of a mammal in need of such **treatment** a permeable membrane having one or more walls, a hollow chamber therewithin, a plurality of holes extending through the walls of the membrane and permitting fluid to enter and exit the chamber of the membrane, each of the holes being sized so that it is large enough to permit inactive Factor VII to enter the chamber of the membrane and activated Factor VIIa to exit the chamber of the membrane but small enough to prevent fibrinogen from entering the chamber of the membrane, a plurality of supports being disposed within the chamber, and an effective amount of a Factor VII activator or a source of the activator being bound to the supports, wherein **inactive factor VII** in **blood** passing through the membrane becomes activated into Factor VIIa upon contact with the activator within the chamber. The present invention also provides a method for treating Hemophilia A or B extracorporeally. The present invention further provides methods for treating AIDS as well as permeable membranes for use in the methods above.

L6 ANSWER 9 OF 20 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 2
AN 2001:332578 BIOSIS
DN PREV200100332578
TI Method for treating hemophilia A and B and AIDS and devices used therein.
AU Pollard, Harvey B. [Inventor]; Pollard, Bette S. [Inventor, Reprint author]
CS 11008 Lamplighter La., Potomac, MD, 20854, USA
PI US 6174299 January 16, 2001
SO Official Gazette of the United States Patent and Trademark Office Patents, (Jan. 16, 2001) Vol. 1242, No. 3. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DT Patent
LA English
ED Entered STN: 11 Jul 2001
Last Updated on STN: 19 Feb 2002
AB The present invention provides a method for treating Hemophilia A or B which comprises implanting in fluid communication with the bloodstream of a mammal in need of such **treatment** a permeable membrane having one or more walls, a hollow chamber therewithin, a plurality of holes extending through the walls of the membrane and permitting fluid to enter and exit the chamber of the membrane, each of the holes being sized so that it is large enough to permit inactive Factor VII to enter the chamber of the membrane and activated Factor VIIa to exit the chamber of the membrane but small enough to prevent fibrinogen from entering the chamber of the membrane, a plurality of supports being disposed within the chamber, and an effective amount of a Factor VII activator or a source of the activator being bound to the supports, wherein **inactive Factor VII** in **blood** passing through the membrane becomes activated into Factor VIIa upon contact with the activator within the chamber. The present invention also provides a method for treating Hemophilia A or B extracorporeally. The present invention further provides methods for treating AIDS as well as permeable membranes for use in the methods above.

L6 ANSWER 10 OF 20 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN DUPLICATE
AN 2000:30807370 BIOTECHNO
TI Technology evaluation: AAV factor IX gene therapy, Avigen Inc
AU Fabb S.A.; Dickson J.G.

CS S.A. Fabb, Department of Biochemistry, Royal Holloway College, University of London, Egham, Surrey TW20 OEX, United Kingdom.
E-mail: s.fabb@rhbnc.ac.uk
SO Current Opinion in Molecular Therapeutics, (2000), 2/5 (601-606), 12 reference(s)
CODEN: CUOTFO ISSN: 1464-8431
DT Journal; General Review
CY United Kingdom
LA English
SL English
AB Gene therapy vectors encoding native and mutant Factor IX sequences for the **treatment** of hemophilia are claimed. **Factor IX** is in the **blood** clotting cascade in humans and is missing or **inactive** in patients with hemophilia B. Recombinant AAV vectors containing the cDNA for Factor IX together with a portion of the intron 1 of this gene are claimed. Various mutant forms of the Factor IX protein such as those which have the ability to bind to human collagen IV are also claimed. The AAV constructs can be injected directly into muscle tissue in at least six sites to achieve their effect. The human Factor IX coding sequences are placed into an AAV vectors under the expression control of the CMV promoter/enhancer. The AAV construct also contains a 1.4 kb fragment of intron 1 of the Factor IX gene. The Factor IX coding sequence is followed by the SV40 polyadenylation sequence and flanked by the AAV ITR sequences. Recombinant virus of 10.sup.1.sup.2 to 10.sup.1.sup.3 genomes/ml was used to inject into mice at a concentration of 10.sup.1.sup.1 or 10.sup.1.sup.0 viral genomes per animal. The injections were in the tibialis anterior and the quadriceps muscle. The human Factor IX was expressed and circulating antibodies were detected. Dogs carrying a mutation in the Factor IX gene which gave them hemophilia B were administered AAV constructs containing dog Factor IX and these showed significantly reduced clotting times.

L6 ANSWER 11 OF 20 IFIPAT COPYRIGHT 2004 IFI on STN DUPLICATE 4
AN 03153227 IFIPAT;IFIUDB;IFICDB
TI METHODS FOR TREATING HEMOPHILIA A AND B AND AIDS AND DEVICES USED THEREIN
INF Pollard; Harvey B., 11008 Lamplighter La., Potomac, MD, 20854
IN Pollard Harvey B
PAF Unassigned
PA Unassigned Or Assigned To Individual (68000)
EXNAM Bockelman, Mark
AG Lambert, Esq., Dennis H.
PI US 5908399 A 19990601
AI US 1996-772034 19960926
XPD 26 Sep 2016
FI US 5908399 19990601
DT Utility; REASSIGNED; EXPIRED
FS MECHANICAL
GRANTED
MRN 009928 MFN: 0849
CLMN 30
GI 9 Drawing Sheet(s), 11 Figure(s).
AB The present invention provides a method for treating Hemophilia A or B which comprises implanting in fluid communication with the bloodstream of a mammal in need of such **treatment** a permeable membrane having one or more walls, a hollow chamber therewithin, a plurality of holes extending through the walls of the membrane and permitting fluid to enter and exit the chamber of the membrane, each of the holes being sized so that it is large enough to permit inactive Factor VII to enter the chamber of the membrane and activated Factor VIIa to exit the chamber of the membrane but small enough to prevent fibrinogen from entering the chamber of the membrane, a plurality of supports being disposed within the chamber, and an effective amount of a Factor VII activator or a source of the activator being bound to the supports, wherein

inactive Factor VII in **blood**
passing through the membrane becomes activated into Factor VIIa upon contact with the activator within the chamber. The present invention also provides a method for treating Hemophilia A or B extracorporeally. The present invention further provides methods for treating AIDS as well as permeable membranes for use in the methods above.

L6 ANSWER 12 OF 20 USPATFULL on STN
AN 1999:151182 USPATFULL
TI Agents affecting thrombosis and hemostasis
IN Wolf, David L., Palo Alto, CA, United States
Sinha, Uma, San Francisco, CA, United States
PA COR Therapeutics Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5990079 19991123
AI US 1998-16400 19980130 (9)
RLI Continuation of Ser. No. US 1995-469301, filed on 6 Jun 1995, now patented, Pat. No. US 5837679 which is a division of Ser. No. US 1994-268003, filed on 29 Jun 1994, now patented, Pat. No. US 5583107 which is a continuation-in-part of Ser. No. US 1994-249777, filed on 26 May 1994, now patented, Pat. No. US 5597799 which is a continuation of Ser. No. US 1991-808329, filed on 16 Dec 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-578646, filed on 4 Sep 1990, now patented, Pat. No. US 5278144
DT Utility
FS Granted
EXNAM Primary Examiner: Degen, Nancy
LREP Morgan, Lewis & Bockius LLP
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 24 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 1981
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Analogs of blood factors which are transiently inactive are useful in **treatment** of diseases characterized by thrombosis. In addition, modified forms of activated blood factors that generate the active blood factor in serum but have extended half-lives are useful in treating hemophilia conditions. These modified forms of the blood factor may be acylated forms which are slowly deacylated in vivo.

L6 ANSWER 13 OF 20 USPATFULL on STN
AN 1999:128656 USPATFULL
TI Factor IX -- polymeric conjugates
IN Hallahan, Terrence W., 82 Hazelwood Ave., Metuchen, NJ, United States 08840
Gilbert, Carl W., 26 Hampton Ct., Basking Ridge, NJ, United States 07920
PI US 5969040 19991019
AI US 1996-766288 19961213 (8)
RLI Division of Ser. No. US 1993-73531, filed on 8 Jun 1993, now patented, Pat. No. US 5621039
DT Utility
FS Granted
EXNAM Primary Examiner: Nutter, Nathan M.
LREP Galgano & Burke
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 526
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Conjugates containing a substance with coagulant activity, such as recombinant Factor IX, non-antigenic polymers, such as poly(ethylene glycol), are disclosed. Also disclosed are methods of forming the novel

conjugates of this invention.

L6 ANSWER 14 OF 20 USPATFULL on STN
AN 1999:128513 USPATFULL
TI Agents affecting thrombosis and hemostasis
IN Wolf, David L., Palo Alto, CA, United States
Sinha, Uma, San Francisco, CA, United States
PA COR Therapeutics, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5968897 19991019
AI US 1998-16403 19980130 (9)
RLI Continuation of Ser. No. US 1995-469301, filed on 6 Jun 1995, now patented, Pat. No. US 5837679 which is a division of Ser. No. US 1994-268003, filed on 29 Jun 1994, now patented, Pat. No. US 5583107 which is a continuation-in-part of Ser. No. US 1994-249777, filed on 26 May 1994, now patented, Pat. No. US 5597799 which is a continuation of Ser. No. US 1991-808329, filed on 16 Dec 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-578646, filed on 4 Sep 1990, now patented, Pat. No. US 5278144
DT Utility
FS Granted
EXNAM Primary Examiner: Degen, Nancy
LREP Morgan, Lewis & Bockius LLP
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 24 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 1908
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Analogs of blood factors which are transiently inactive are useful in **treatment** of diseases characterized by thrombosis. In addition, modified forms of activated blood factors that generate the active blood factor in serum but have extended half-lives are useful in treating hemophilic conditions. These modified forms of the blood factor may be acylated forms which are slowly deacylated in vivo.

L6 ANSWER 15 OF 20 USPATFULL on STN
AN 1998:144079 USPATFULL
TI Agents affecting thrombosis and hemostasis
IN Wolf, David L., Palo Alto, CA, United States
Sinha, Uma, San Francisco, CA, United States
PA COR Therapeutics, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5837679 19981117
AI US 1995-469301 19950606 (8)
RLI Division of Ser. No. US 1994-268003, filed on 29 Jun 1994, now patented, Pat. No. US 5583107 which is a continuation-in-part of Ser. No. US 1994-249777, filed on 26 May 1994, now patented, Pat. No. US 5597799 which is a continuation of Ser. No. US -808329 which is a continuation-in-part of Ser. No. US 1990-578646, filed on 4 Sep 1990, now patented, Pat. No. US 5278144
DT Utility
FS Granted
EXNAM Primary Examiner: Fleisher, Mindy; Assistant Examiner: Degen, Nancy J.
LREP Morrison & Foerster LLP
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 2092
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Analogs of blood factors which are transiently inactive are useful in **treatment** of diseases characterized by thrombosis. In addition, modified forms of activated blood factors that generate the active blood factor in serum but have extended half-lives are useful in treating

hemophilic conditions. These modified forms of the blood factor may be acylated forms which are slowly deacylated in vivo.

L6 ANSWER 16 OF 20 USPATFULL on STN
AN 97:31760 USPATFULL
TI Factor IX- polymeric conjugates
IN Hallahan, Terrence W., 82 Hazelwood Ave., Metuchen, NJ, United States 08840
Gilbert, Carl W., 26 Hampton Ct., Basking Ridge, NJ, United States 07920
PI US 5621039 19970415
AI US 1993-73531 19930608 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Mullis, Jeffrey C.
LREP Galgano & Burke
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 636
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Conjugates containing a substance with coagulant activity, such as recombinant Factor IX, non-antigenic polymers, such as poly(ethylene glycol), are disclosed. Also disclosed are methods of forming the novel conjugates of this invention.

L6 ANSWER 17 OF 20 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 5
AN 1997:744 CAPLUS
DN 126:84595
TI Agents affecting thrombosis and hemostasis
IN Wolf, David L.; Sinha, Uma
PA Cor Therapeutics, Inc., USA
SO U.S., 33 pp., Cont.-in-part of U.S. Ser. No. 249,777.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5583107	A	19961210	US 1994-268003	19940629
	US 5278144	A	19940111	US 1990-578646	19900904
	JP 2002234899	A2	20020823	JP 2001-395485	19910904
	US 5597799	A	19970128	US 1994-249777	19940526
	US 5635481	A	19970603	US 1995-467339	19950606
	US 5650314	A	19970722	US 1995-470807	19950606
	US 5837679	A	19981117	US 1995-469301	19950606
	US 5795863	A	19980818	US 1995-487037	19950607
	CA 2190642	AA	19960111	CA 1995-2190642	19950628
	WO 9600577	A1	19960111	WO 1995-US8368	19950628
	W: AU, CA, JP, MX				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9529585	A1	19960125	AU 1995-29585	19950628
	AU 712271	B2	19991104		
	EP 766563	A1	19970409	EP 1995-925461	19950628
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 10502351	T2	19980303	JP 1996-503483	19950628
	US 5968897	A	19991019	US 1998-16403	19980130
	US 5990079	A	19991123	US 1998-16400	19980130
	US 2004072757	A1	20040415	US 2003-712332	20031113
PRAI	US 1990-578646	A2	19900904		
	US 1991-808329	B1	19911216		
	US 1994-249777	A2	19940526		
	JP 1991-517527	A3	19910904		

US 1994-268003	A3	19940629	
US 1995-469301	A1	19950606	
WO 1995-US8368	W	19950628	
US 1998-16403	A1	19980130	
US 1999-362207	B1	19990728	
US 2000-671346	A1	20000927	

AB Analogs of **blood** factors, such as **factor X**, which are transiently **inactive** are useful in **treatment** of diseases characterized by thrombosis. In addition, modified forms of activated blood factors that generate the active blood factor in serum but have extended half-lives are useful in treating hemophilic conditions. These modified forms of the blood factor may be acylated forms which are slowly deacylated in vivo.

L6 ANSWER 18 OF 20 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1988:118953 CAPLUS
DN 108:118953
TI Purification of blood coaguation factor IX for **treatment** of hemophilia
IN Nishimaki, Hideo; Kameyama, Matsuhisa; Nakamura, Yukihiko; Iga, Yoshiro; Suyama, Tadakazu
PA Green Cross Corp., Japan
SO Jpn. Kokai Tokkyo Koho, 4 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 62201824	A2	19870905	JP 1986-44941	19860228
PRAI JP 1986-44941		19860228		

AB **Inactive blood** coagulation **factor IX** (I) is prepared free of active blood coagulation factors II, VII, IX, and X. Normally, I is a mixture of blood coagulation factors II, VII, IX, and X in active forms as well as in inactive forms, and the active factors are not desirable in clin. use, and, therefore, eliminated by treating I with insol. materials containing immobilized aminobenzamidine, aminophenylguanidine, or basic amino acids. I consisting of blood coagulation factor IX 1044, factor II 1166, factor VII 222, and factor X 492 units were dissolved in 40 mL of 1.5% by weight/volume NaCl-0.5% by weight/volume Na citrate (pH 7.0) and mixed with 2 g of benzamidine-sepharose 6B which had been equilibrated with the same solution The mixture was stirred at 4° for 1 h and centrifuged. The supernatant contained I free of active coagulation factors.

L6 ANSWER 19 OF 20 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1976:537224 CAPLUS
DN 85:137224
TI Proteins induced by vitamin K antagonists (PIVKAs)
AU Lindhout, M. J.; Kop-Klaassen, B. H. M.
CS Dep. Biochem., Maastricht Med. Fac., Maastricht, Neth.
SO Boerhaave Series for Postgraduate Medical Education (1975), 10(Prothrombin Relat. Coagulation Factors), 274-88
CODEN: BSPEDP; ISSN: 0304-9167
DT Journal
LA English
AB Using immunodiffusion tests, blood coagulation factor IX [9001-28-9] and **blood** coagulation **factor X** [9001-29-0] had common antigenic determinants with the **inactive** precursors, PIVKA-IX and PIVKA-X, which are induced by vitamin K antagonists. However, the PIVKA's had a lower affinity for Al(OH)3 than factors IX and X, a phenomenon probably related to the lack of Ca-binding sites on the PIVKA's. The normal blood coagulation factor VII [9001-25-6], factor IX,

and factor X were completely absorbed onto Al(OH)3, whereas 40% of blood coagulation factor II [9001-26-7] activity remained in the blood. In cows treated with phenprocoumon [435-97-2] (600 mg the first day and 200 mg/day, thereafter), plasma factor X activity was decreased, which resulted in the appearance of PIVKA-X two-dimensional immunoelectrophoresis in the presence of Ca; the presence of both factor X and PIVKA-X after anticoagulant **treatment** was also demonstrated. The existence of PIVKA-II and PIVKA-IX were also demonstrated by this method. Studies with PIVKA-X indicated that its active site was intact when compared to factor X, but that there was in addition to or as a consequence of the lack of Ca binding sites, another defect, resulting in a lower rate of activation of prothrombin by PIVKA-X.

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AU Lenstra, R., et al
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CSR MID-ATLANTIC/MIDDLE ATLANTIC REGION; UNITED STATES OF AMERICA; NORTH AMERICA
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L Number	Hits	Search Text	DB	Time stamp
1	715	clotting near3 (factor adj (II or VII or IX or X))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/31 15:44
2	106	L1 near6 ((dominant negative) or inactive)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/31 15:43
3	0	(L1 near6 ((dominant negative) or inactive)) near10 (treat*)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/31 15:43
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7	1910	blood near10 (factor adj (II or VII or IX or X))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/31 15:45
8	104	((dominant adj negative) or inactive) near6 (factor adj (II or VII or IX or X))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/31 15:46
9	0	(blood near10 (factor adj (II or VII or IX or X))) and (((dominant adj negative) or inactive) near6 (factor adj (II or VII or IX or X))) and treat*	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/31 15:46

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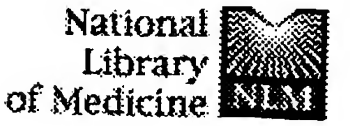

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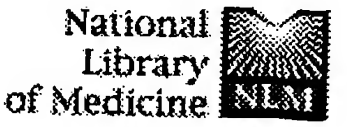

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